1. (Amended) A method for identifying a subject at risk of developing a cancer characterized by abnormally increased methylation of a CpG island containing TMS1 nucleic acid molecule comprising

determining a level of methylation of a CpG island containing TMS1 nucleic acid molecule in a biological sample from a subject, and

comparing the level of methylation of the CpG island containing TMS1 nucleic acid molecule in the biological sample to a control

wherein the CpG island containing TMS1 nucleic acid molecule is selected from the group consisting of

(a) nucleic acid molecules which hybridize under stringent conditions to a complement of a molecule consisting of SEQ ID NO:4 and which code for a native TMS1 polypeptide, and

(b) complements of (a), and

wherein an increase in the level of methylation of the CpG island containing TMS1 nucleic acid molecule in the biological sample compared to the control identifies a subject at risk of developing the cancer.

47. (Amended) A method for identifying a subject having cancer who is at risk of being non-responsive to an apoptosis-dependent anti-cancer therapy comprising:

determining a level of methylation of a CpG island containing TMS1 nucleic acid molecule in a biological sample from a subject having cancer, and

comparing the level of methylation of the CpG island containing TMS1 nucleic acid molecule in the biological sample to a control,

wherein the CpG island containing TMS1 nucleic acid molecule is selected from the group consisting of

(a) nucleic acid molecules which hybridize under stringent conditions to a complement of a molecule consisting of SEQ ID NO:4 and which code for a native TMS1 polypeptide, and

(b) complements of (a), and

wherein an increase in the level of methylation of the CpG island containing TMS1 nucleic acid molecule in the biological sample compared to the control identifies a subject who is at risk of being non-responsive to an apoptosis-dependent anti-cancer therapy.

110. The method of claim 1, wherein the level of methylation is determined using a technique selected from the group consisting of methylation sensitive restriction analysis,

methylation specific polymerase chain reaction (MSP), sequencing of bisulfite modified DNA, methylation-sensitive single nucleotide primer extension (Ms-SNuPE), and combined bisulfite restriction analysis (COBRA).

111. The method of claim 1, wherein the biological sample is breast tissue.

112. The method of claim 1, wherein the control comprises a normal tissue from a normal subject.

113. The method of claim 47, wherein the level of methylation is determined using a technique selected from the group consisting of methylation sensitive restriction analysis, methylation specific polymerase chain reaction (MSP), sequencing of bisulfite modified DNA, methylation-sensitive single nucleotide primer extension (Ms-SNuPE), and combined bisulfite restriction analysis (COBRA).

114. The method of claim 47, wherein the cancer is breast cancer.

115. The method of claim 113, wherein the biological sample is a breast cancer tumor.

116. The method of claim 47, wherein the control is normal tissue from a normal subject.

117. The method of claim 116, wherein the control is normal tissue from the subject having cancer.

118. (Amended) The method of claim 47, wherein the apoptosis-dependent anti-cancer therapy is a DNA damaging anti-cancer therapy.

119. (Amended) The method of claim 47, wherein the apoptosis-dependent anti-cancer therapy is radiation therapy.

120. (Amended) The method of claim 47, wherein the apoptosis-dependent anti-cancer therapy is chemotherapy.

121. (Amended) The method of claim 47, further comprising administering to the subject at risk of being non-responsive to an apoptosis-dependent anti-cancer therapy, a demethylating agent and an apoptosis-dependent anti-cancer therapy.

122. (Amended) The method of claim 47, further comprising administering to the subject at risk of being non-responsive to an apoptosis-dependent anti-cancer therapy, an anti-cancer therapy selected from the group consisting of biological response modifying therapy, immunotherapy, cancer vaccine therapy, hormone therapy and angiogenesis inhibiting therapy.

123. (New) A method for identifying a subject at risk of developing a cancer characterized by abnormally increased methylation of a nucleic acid molecule comprising a TMS1 CpG island comprising

determining a level of methylation of a nucleic acid molecule comprising a TMS1 CpG island in a biological sample from a subject, and

comparing the level of methylation of the nucleic acid molecule comprising a TMS1 CpG island in the biological sample to a control

wherein the nucleic acid molecule comprising a TMS1 CpG island is selected from the group consisting of

(a) nucleic acid molecules which hybridize under stringent conditions to a complement of a molecule consisting of SEQ ID NO:4, and

(b) complements of (a), and

wherein an increase in the level of methylation of the nucleic acid molecule comprising a TMS1 CpG island in the biological sample compared to the control identifies a subject at risk of developing the cancer.

124. (New) A method for identifying a subject having cancer who is at risk of being non-responsive to an apoptosis-dependent anti-cancer therapy comprising:

determining a level of methylation of a nucleic acid molecule comprising a TMS1 CpG island in a biological sample from a subject having cancer, and

comparing the level of methylation of the nucleic acid molecule comprising a TMS1 CpG island in the biological sample to a control,